attitude of society, his friends, relatives, associates, and even casual contacts influences the response of the man more than is usually appreciated.

Furthermore, one of the most important human beings in the world of the patient is his doctor. The reconstructive surgeon whether he be repairing a mangled hand or improving a scarred face—whether he be striving for restoration of function or appearance—endeavours constantly to keep in the forefront of his consciousness the total personality entrusted to his care. No longer does the expert but unimaginative technician reach the peak in plastic surgery. His ministrations must succeed not only surgically but also psychologically. It is easy to transfer skin from the thigh to the hand or from the arm to the face and to have a 100% take. It is not so easy to restore the patient to his place in the social or economic world. In truth the mere fact that the surgeon has outlined for him a long series of multiple stage operations running into years, tends to convince the tremulous victim that he must be horrible and that he must hide from his fellow man until his kindly benefactor, the surgeon, has made him fit to appear.

The surgeon must ever be able to support his patient until the thinking and feeling of society be moulded to the point where scars of the body even though not exalted as necessary emblems of valour are at least regarded in their proper perspective and are seen to be of little importance in the structure of the real personality.

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RÉSUMÉ

Le chirurgien qui s'occupe de chirurgie plastique doit être, non seulement un expert dans son art, mais aussi un psychologue averti. Il est le mieux placé pour préparer son malade aux éventualités de l'attitude du public. Il faut lutter contre cette habitude qu'ont certains d'associer les cicatrices avec quelque chose d'horrible. Il faut bien savoir que ces cicatrices n'ont pas été voulues et qu'elles sont plus anoblissantes que dégradantes. Le mutilé n'a pas subi de mutilation de sa personnalité. Il faut s'habituer à voir les cicatrices à leur véritable échelle, ou mieux encore, à les ignorer. On n'évalue pas la personnalité par les signes extérieurs. La chirurgie plastique a beaucoup fait mais elle ne peut pas tout restaurer. Notre charité et notre bienveillance devront accomplir aux yeux du mutilé la complète restauration. JEAN SAUCIER

THE VALUE OF SPLENECTOMY IN HÆMATOLOGICAL DISORDERS

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SPLENECTOMY is now recognized as a valuable therapeutic measure in certain diseases of the blood and blood-forming organs. The veil of mystery enshrouding the spleen is gradually being lifted by hæmatological research. The present concepts of splenic function and the newer methods of diagnosis provide a more rational basis for the operation, while the improvement in surgical technique and care of the patient should lessen its dangers. I shall refer briefly to some of the hæmatological conditions in which splenectomy may be valuable, and give some illustrative case reports.

HÆMOLYTIC ANÆMIA

This large group includes some conditions which are cured or benefited by splenectomy, and others in which the operation is contraindicated. Congenital hæmolytic jaundice, according to Dameshek[1] is probably due to hypersplenism, but whether the spleen exerts only a hæmolytic effect, or also influences the maturation of the red cells and their delivery from the marrow has not been settled. Mild cases may live to an advanced age more or less symptom-free, but in the majority chronic ill-health is the rule, with the ever-present danger of a hæmolytic crisis or some serious complication. Splenectomy is followed by uniformly beneficial and lasting results. Operating during a crisis is risky but may be life-saving. Transfusions may occasion severe reactions. After splenectomy the increased fragility of the red cells and spherocytosis may persist, but the patients are greatly improved in health and the prognosis is excellent.

Acquired hæmolytic jaundice is a more serious condition. Splenectomy may be the only course but the risk is greater than in the congenital type. In the acute form Dameshek and Schwartz[4] reported it curative in 20 out of 23 cases, and advocated its use when transfusions gave temporary benefit only. In Cooley's anæmia and sickle-cell anæmia the operation may give symptomatic relief and in some instances may retard the progress of the disease. It is contraindicated in the hæmolytic anæmia associated with Hodgkin's disease,

lymphosarcoma or lymphatic leukæmia. Sharpe and Tollman[5] report the operation a failure in refractory hæmolytic anæmia. Stransky and Regala[6] found that in atypical familial hæmolytic anæmia splenectomy was followed by erythroblastic anæmia.

CASE 1

H.L.S., male, aged 30. Admitted to hospital in November, 1942, complaining of nausea, vomiting, loss of appetite, tired feeling and jaundice. Family history, negative for any blood dyscrasias, jaundice, etc. Personal history, O.D.C. tonsillectomy, 1932 with two postoperative hæmorrhages. During convalescence he contracted scarlet fever. Present illness: The symptoms began about one week before admission. There was a definite icterus, but no hæmorrhages or purpura. Spleen and liver were not palpable. Remainder of examination was negative. Icterus index 44; Hb. 60%; red blood cells 3,710,000; colour index 0.80; white blood cells 11,700; reticulocytes 17%. Fragility of the red cells increased. Urine, negative. A diagnosis of hæmolytic anæmia was made. Patient recovered from the hæmolytic crisis and during the next two years slight jaundice was present. In 1944 the spleen became palpable and gradually increased in size until 1946 when it was about 3″ below the costal margin. The patient suffered considerable abdominal discomfort, nervousness and tendency to fatigue. Icterus index ranged between 30 and 40.

In July, 1947, physical examination revealed a moderate icterus and splenomegaly, but was otherwise negative. Urine was negative for bile. Hb. 80%; red blood cells 4,140,000; white blood cells 11,800; reticulocytes 19%. The red cells showed some achromia, anisocytosis and microspherocytosis, with some polychromatophilia and a few normoblasts. Fragility began at 0.50 and was complete at 0.42. The sternal marrow showed marked increase in activity. Liver function tests, negative. On July 30, the spleen was removed. The patient made an excellent recovery. The jaundice, abdominal discomfort and fatigue soon disappeared. At the present time he feels better than he has for many years. Hb. 102%; red blood cells 5,180,000; white blood cells 10,200; reticulocytes 1%; icterus index 4.

It is difficult to state whether this is a case of congenital or acquired hæmolytic anæmia, but splenectomy was definitely indicated and unquestionably beneficial.

THROMBOCYTOPENIC PURPURA

Dameshek[1] distinguishes four types of this disease. (1) Due to bone marrow disease in which the megakaryocytes are diminished as part of a generalized disturbance, as in aplasia, leukæmia, etc. (2) Due to a selective bone marrow disturbance from toxic, chemical or allergic cause. (3) Due to hypersplenic effects of a splenomegaly. (4) Idiopathic, in which an active substance from the spleen causes an inhibition of growth and delivery of platelets from the megakaryocytes to the blood.

In considering splenectomy for thrombocytopenic purpura it should be borne in mind that the condition may respond to medical measures. Postponement of the operation, however, may be regretted in the event of hæmorrhage into vital structures such as the brain or the eye. The risk of splenectomy may be preferable to a wait-and-see policy. The chief value of the operation is in the idiopathic type or in those cases secondary to hypersplenism.

CASE 2

Mr. E.C., aged 20 years was admitted to hospital August 22, 1946, on account of injuries sustained in a motor accident the previous day.

Family history, unimportant.

In 1943 he injured his knee and there was a large hæmorrhage into the joint. A few days later he had a severe epistaxis, and one month later hæmorrhage in the left eye. Vision in this eye had remained impaired with occasional diplopia. Since 1943 he had manifested a tendency to bleed easily following trauma. He had numerous epistaxes and attacks of purpura. The last epistaxis was in April, 1946. An appendectomy in 1946 was uncomplicated. Physical examination on admission revealed contusions of the face, head, chest, arms, left thigh and over the sacrum. There were no petechiæ present. Spleen and liver were not palpable. Vision in the left eye was impaired, with muscle imbalance and a mild retinitis. Remainder of examination was negative. X-rays disclosed a linear fracture of the left frontal region without depression.

Urine, normal; Kahn, negative; Hgb. 70%; bleeding time, over 20 minutes; coagulation time, 3½ minutes; prothrombin time, 18 seconds; clot retraction, 24 hours; platelets, 45,100; tourniquet test, positive.

The diagnoses were, fracture of the skull, multiple contusions and thrombocytopenic purpura. Recovery from the accident was satisfactory, no further bleeding occurred and the patient was discharged on September 10 with instructions to return in one month. He was readmitted on October 11. Since leaving hospital he had had no hæmorrhages, but had noticed a few purpuric spots on his limbs. Physical examination revealed a few ecchymoses in the skin. The spleen was not palpable. General physical condition was good.

Urine, normal; Hgb. 75%; red blood cells 3,850,000; white blood cells 10,900. The red cells showed hypochromia, anisocytosis and a predominance of microcytes. No abnormal white cells; juveniles 0; bands 1; seg. polymorphonuclears 49; lymphocytes 45; large monocytes 1; eosinophiles 2; basophiles 2. Blood platelets 19,250; bleeding time over 10 minutes; coagulation time 3½ minutes; clot retraction 20 hours; tourniquet test positive. On October 22 splenectomy by Dr. Kinley. The spleen was large and soft and the splenic veins greatly dilated.

Pathologist's report.—Spleen weighed 370 gm., dark red, soft and congested; no fibrosis or miliary tubercles visible. There were numerous tubercle-like follicles without caseation in the centres of most of the Malpighian bodies. Numerous epithelioid cells and multi-nucleated giant-cells with the reticulum still largely present in them. The diagnosis rests between sarcoidosis and miliary tuberculosis, but I rather favour the former. The spleen otherwise shows congestion of its sinuses, but no excess of megakaryocytes or eosinophiles. There are some polymorphonuclears such as one usually sees in a typical case of thrombocytopenic purpura (Dr. R. P. Smith).

The postoperative course was excellent. On October 29, the blood picture was as follows: Hb. 92%; red blood cells 4,880,000; white blood cells 15,100; juveniles 1; bands 4; seg. polymorphonuclears 75; lymphocytes 16; large monocytes 2; eosinophiles 2; basophiles 0. Plasma proteins 7.8 gm. %; albumin 4.44; globulin 3.36. Volmer patch test and intracutaneous tuberculin test, negative.

The patient was discharged in excellent condition on November 13, 1946. In view of the pathological findings, the high globulin, and the negative evidence of tuberculosis, it was felt that this was a case of sarcoid spleen with secondary thrombocytopenic purpura. There was no other evidence of sarcoidosis. The patient reported on November 7, 1947, that he has been in splendid

health since the operation. He has gained weight and has had only two slight nose-bleeds. Blood picture: Hb. 110%; red blood cells 5,500,000; white blood cells 20,050; platelets 110,000; bleeding time normal; plasma proteins 7.1; albumin 5.4; globulin 1.7.

PRIMARY SPLENIC NEUTROPENIA

This condition was first described by Frank[7] in 1916 as "Aleukia Splenica". Reissmann[8] reported the first case of splenic neutropenia associated with splenomegaly and cured by removal of the spleen. Wiseman and Doan[9] in 1939 reported 3 cases which they designated primary splenic neutropenia and cured by splenectomy. The characteristic features were splenomegaly, granulocytopenia of the peripheral blood and myeloid hyperplasia of the bone marrow. In a subsequent report in 1942[10] they describe acute, sub-acute and chronic forms, and pointed out that the syndrome was closely related to congenital hæmolytic jaundice and essential thrombocytopenic purpura, as in some cases the neutropenia was accompanied by hæmolytic anæmia or thrombocytopenia or both. Simultaneous reduction of all cellular elements is called splenic pancytopenia. Excessive splenic phagocytosis was regarded as the responsible mechanism. Dameshek, as had Reissmann, suggested that the neutropenia was due to a hormonal splenic influence on the bone marrow, that is, a form of hypersplenism.

Whether the condition is due to splenic phagocytosis or to hormonal splenic influence on the marrow, has not been decided. In all cases reported to date splenomegaly was present and splenectomy resulted in a prompt and sustained remission of the neutropenia. Hattersley[11] recently reported a case of chronic neutropenia without splenomegaly which failed to respond to the operation, and suggested that lack of splenomegaly should be regarded as a contraindication. According to Dameshek[1] splenic neutropenia may be primary, or it may be associated with the splenomegaly of Felty's syndrome, Boeck's sarcoid, Gaucher's disease, tuberculosis of the spleen, malaria or kala-azar.

CASE 3

J.B., male aged 12 years, an only child of a neurotic and rather anæmic mother, had the ordinary diseases of childhood and diphtheria. He was subject to epistaxis. In December, 1945, he had influenza and in February, 1946, a severe attack of varicella. Convalescence was slow and during the next few months the patient suffered from weakness, anorexia and a tendency to perspire easily. On August 6, 1946, he was found by his family physician to have a temperature of 101° and he was pale and tired looking. Blood count revealed a marked anæmia, and a leucocyte count of 1,200. (Details of count not available, but granulocytes were very low.) Agranulocytosis was diagnosed. Penicillin and vitamin therapy were started. Fever persisted for several days, and, for a few weeks vomiting and diarrhœa were troublesome. The spleen and liver were not palpable and there were no enlarged glands. At this time aleukæmic leukæmia was suspected. A blood transfusion was given on August 24, and liver, iron and vitamins daily. Considerable improvement followed. In September the patient was brought to Halifax for a sternal marrow examination and hæmogram. The findings were characteristic of splenic neutropenia and splenectomy was advised.

The patient was admitted to hospital on November 15, 1946. He had no complaints other than some weakness. The temperature and pulse rate were very slightly elevated. Physical examination revealed a moderate obesity, pallor and some enlargement of the spleen to percussion. A few enlarged glands were present in the submaxillary regions. Examination was otherwise negative.

Laboratory findings.—Urine, normal; Kahn, negative; bleeding and coagulation times, normal; Widal and skin test for undulant fever, negative; Hgb. 72%; red blood cells 3,340,000; colour index 1.09; white blood cells 2,200; platelets 260,000. Mild anisocytosis, no polychromasia or nucleated red cells; no immature white cells. Differential: juvenile 0; band 0; seg. polymorphonuclears 13; lymphocytes 54; large mononuclears 28; eosinophiles 2; basophiles 2; plasma cell 1. Comment: neutropenia with relative lymphocytosis and monocytosis and a moderate normocytic anæmia.

The marrow from a sternal puncture showed a moderate myeloid hyperplasia with maturation arrest at the band form stage. The polymorphonuclears were 2.0% and eosinophiles 1.5%. The appearances did not suggest leukæmia but in conjunction with the marked peripheral neutropenia were in keeping with the diagnosis of primary splenic neutropenia.

On November 28, splenectomy was performed by Dr. C. E. Kinley. The organ was moderately enlarged and a few adhesions were present. A small accessory spleen was seen near the hilum and this was also removed. During the operation the patient received 500 c.c. of saline and 500 c.c. of blood. Leucocyte counts were performed before, during and after the operation with the following results.

TABLE I.

Time	*Leucocytes*
Preoperative	2,800
After handling spleen	5,650
25 minutes after clamping pedicle	6,150
45 minutes after clamping pedicle	8,300
4 hours after clamping pedicle	10,100
7 hours postoperative	14,450
13 hours postoperative	9,500
24 hours postoperative	9,300
December 3, 1946	6,600
December 13, 1946	5,800

DIFFERENTIAL SCHILLING COUNTS

Cells	*Pre-operative*	*4 hours post-operative*	*7 hours*	*13 hours*	*24 hours*	*Dec. 3*	*Dec. 13*
	%	%	%	%	%	%	%
Myelocytes	0.0	0.0	1.0	0.0	0.0	0.0	0.0
Juveniles	0.0	1.0	1.0	2.0	0.0	0.0	2.0
Band forms	1.0	45.0	35.0	22.0	15.0	0.0	1.0
Seg. polymorpho-nuclears	2.0	20.0	48.0	48.0	53.0	20.0	23.0
Lymphocytes	79.0	14.0	5.0	9.0	13.0	57.0	62.0
Large monocytes	16.0	21.0	10.0	19.0	18.0	18.5	9.0
Eosinophiles	1.0	0.0	0.0	0.0	0.0	3.0	1.0
Basophiles	1.0	0.0	0.0	0.0	1.0	1.0	2.0

Pathologist's report.—The spleen is enlarged, weighing 575 gm. Its surface is smooth and a deep purple colour. On section the cut surface is very congested, free blood pouring from it. The Malpighian bodies are very prominent, standing out as whitish nodules varying from 1 to 2 mm. in diameter. Histology reveals marked congestion of the sinusoids of the pulp with very marked hyperplasia of the lymph follicles. There is some endothelial cell hyperplasia but no active phagocytosis of blood cells. I can detect no evidence of malignancy, tuberculosis or sarcoidosis. Diagnosis, non-specific lymphoid hyperplasia (Dr. R. P. Smith).

The postoperative course was uneventful and the patient was discharged on December 14, 1946. Since that time he has been receiving iron and vitamin therapy. He has been in good health, carrying on normal activities. Blood pictures have been done at three month intervals and both are within normal limits, except that the total white cell count remains elevated at 10,700 and 13,400, but the polymorphonuclear count is somewhat low at 36 and 34%. This may be due to an accessory spleen which is still exerting a mild hypersplenic influence.

FELTY'S SYNDROME

Felty[12] in 1924 reported 5 cases of adults with polyarthritis of the atrophic variety, fever, secondary anæmia, leukopenia, splenomegaly and tachycardia. Hanrahan and Miller[13] were the first to report the beneficial effects of splenectomy in the condition. Steinberg[14] studied the bone marrow in Felty's syndrome and in other forms of atrophic arthritis and found that all showed hyperplasia. In his opinion the neutropenia was due to a splenic influence acting as a barrier to the releasing of the granulocytes from the marrow into the blood as demonstrated by the improvement following splenectomy. Felty's syndrome may be regarded as an example of splenic neutropenia due to hypersplenism but differing from the primary form. It is generally agreed that splenectomy will not likely influence the arthritis, but in patients with a steadily enlarging spleen or in those subject to intercurrent infections the operation seems justified.

CASE 4

Mrs. M.N., aged 71 years. Admitted to hospital August 23, 1946, complaining of (1) a mass in the left side of the abdomen; (2) arthritis. Family history, not contributory. Personal history, O.D.C. Scarlet fever. Pneumonia 1945. Ten children; one miscarriage. Polyarthritis had been present for 5 years. About March, 1946 the patient discovered a mass in the upper part of the abdomen in the left side, which had gradually increased in size, causing discomfort but no other symptoms. She was a thin elderly woman with rheumatoid arthritis, marked splenomegaly and slight hepatomegaly. The examination was otherwise essentially negative.

Laboratory examination.—Urine and blood-chemistry, normal; Kahn, negative; bleeding time, 20 minutes; coagulation time, 2¾ minutes; Hgb. 72%; red blood cells 4,490,000; colour index 0.8; white blood cells 2,400; platelets 179,000. Differential: juvenile 0; band 2.0; seg. polymorphonuclears 0.0; lymphocytes 81.0; large monocytes 12.0; eosinophiles 5; basophiles 0. Normocytic anæmia with slight polychromasia; no immature white cells. Profound neutropenia present with only 48 granulocytes per c.mm. of blood.

TABLE II.

STERNAL PUNCTURE MYELOGRAM

	Percentage
Myeloblasts	3.0
Promyelocytes	8.0
Myelocytes	16.5
Juveniles	16.5
Band forms	13.5
Polymorphonuclears	1.5
Eosinophiles	1.5
Basophiles	0.0
Lymphocytes	14.0
Monocytes	0.0
Erythroblasts	8.0
Early normoblasts	7.0
Late normoblasts	10.5

The myelogram shows a moderate myeloid hyperplasia with maturation arrest at the band form stage. There is evidence of active erythropoiesis. The appearances do not suggest a myelogenous leukæmia.

The association of rheumatoid arthritis, splenomegaly, granulocytopenia with myeloid hyperplasia of the marrow justified a diagnosis of Felty's syndrome. In view of the discomfort occasioned by the spleen which had been gradually increasing in size, and with the granulocytopenia rendering the patient liable to infections, splenectomy was advised with the realization that the arthritis would not likely be benefited. On September 20, 1946, splenectomy was performed by Dr. Kinley. The massive spleen presented numerous adhesions and old infarcts. The splenic artery was markedly dilated, and there was a large vein in the gastro-splenic ligament. Pathologist's report: Spleen is much enlarged weighing 750 gm. The surface is smooth and of a grayish purple colour. At the upper pole there is an irregular area of chronic perisplenitis with two depressed stellate scars. Lying in the hilum are three accessory spleens, the size of marbles. Cut

surface is firm, glistening and deep red in colour. The Malpighian bodies stand out clearly but are not unduly hyperplastic in the gross. Histological examination, shows some chronic perisplenitis with a marked diffuse endothelial hyperplasia, the endothelial cells showing definite phagocytosis of polymorphonuclears and a few red cells. Such an appearance is characteristic of a chronic neutropenic splenomegaly. The biopsy of the liver only shows some fatty degeneration and a little lymphocytic infiltration of the portal tract areas. There is no special proliferation of the Kupffer cells (Dr. R. P. Smith).

Following the operation the patient was given penicillin and a blood transfusion. The postoperative course was normal.

TABLE III.

EFFECT OF SPLENECTOMY ON THE LEUCOCYTE COUNT

	White blood count
September 20—Immediately before splenectomy	1,500
15 minutes after pedicle clamped	7,850
30 " " " "	4,300
6 hours " " "	16,650
11 " " " "	18,500
September 21—24 " " " "	22,350
September 23	17,700
September 25	12,750
October 7	5,900

The patient was discharged from hospital October 8, 1946. Her general health has been much better since the operation, even the arthritis being less troublesome. She is able to do some housework whereas previously she was almost a complete invalid. The blood picture on September 22, 1947, was as follows: Hb. 73%; red blood cells 4,330,000; white blood cells 10,200; juvenile 0; bands 1; seg. polymorphonuclears 46; lymphocytes 52; large monocytes 0; eosinophiles 1; basophiles 0.

The marked improvement in this case of Felty's syndrome demonstrates the possibilities of splenectomy for the condition.

BANTI'S SYNDROME

In 1883 Banti first called attention to this condition and subsequently described its three stages, the anæmic, the transitional and the ascitic. Until recently the results of splenectomy have been disappointing. Whipple[15] has reported a most interesting investigation of portal hypertension. This may be due to intrahepatic block, that is cirrhosis, or to extrahepatic block in the portal bed. Whether the block is intra- or extra-hepatic can usually be determined by liver function tests. If the extrahepatic block is distal to the junction of the coronary vein with the portal system, splenectomy will give satisfactory results. In other cases of extrahepatic or in intrahepatic block it should provide some relief until portal hypertension builds up again, but it will not effect a cure. Blakemore and Lord[16] have had encouraging results in the treatment of portal hypertension by establishing porto-caval shunts, but the operation is experimental and difficult. No doubt these new concepts of Banti's syndrome will mean a more careful selection of cases for operation.

CASE 4

Mrs. Z., aged 25. Admitted to hospital April 2, 1945, complaining of anæmia and enlarged spleen. Family history non-contributory. Personal history: jaundice at 7 and again at 17. She had always been pale; no history of hæmorrhages. Splenomegaly was discovered 5 years previously. Obstetrical history—two children, both normal deliveries. Physical examination, moderately pale, well nourished; slight cardiac enlargement with systolic murmurs in the mitral and aortic areas; marked splenomegaly; no enlarged lymph nodes, blood pressure 120/80. Early pregnancy was suspected.

TABLE IV.

DIFFERENTIAL SCHILLING COUNTS

Cells	*September 4, 1946*	*September 20 30 minutes after pedicle clamped*	*September 21*	*September 25*	*October 7*
Myelocytes	0	0	1	0	0
Juveniles	0	0	2	3	0
Band forms	2	10	63	12	0
Seg. polymorphonuclears	0	4	19	62	48
Lymphocytes	81	71	8	15	31
Large monocytes	12	9	7	4	17
Eosinophiles	5	3	0	3	3
Basophiles	0	3	0	1	1

Laboratory findings: Kahn, negative; blood chemistry, normal. Urinalysis, albumin one plus; sugar negative. Microscopically, a few red cells and a few white blood cells; Hb. 53%; red blood cells 2,500,000; white blood cells 5,900; differential count, juvenile 0; band 4; seg. polymorphonuclears 46; lymphocytes 48; large monocytes 1; eosinophiles 1; basophiles 0; microcytes with a few macrocytes; no nucleated reds, but a few show polychromatophilia; platelets 114,000; icterus index 10; Fouchet test, positive; van den Bergh, direct immediate, negative; direct biphasic, negative; direct delayed, positive; indirect, slightly positive. Hæmatocrit, 24 mm. (56% of normal). Volume index 1.1. X-ray examination of œsophagus, negative for varicosities.

Following a transfusion, patient was discharged April 18, 1945. Banti's syndrome was regarded as the most likely diagnosis.

Readmitted June 1, 1945. Examination revealed a three months' pregnancy. Otherwise her condition was unchanged; Hgb. 45%; red blood cells 2,210,000; white blood cells 5,700. Differential count, juvenile 2; band 4; seg. polymorphonuclears 65; lymphocytes 26; large monocytes 1; eosinophiles 2; basophiles 0. Platelets 265,000. Bleeding time 2.2 minutes. Owing to the large size of the spleen and the likelihood of this complicating

pregnancy, it was felt that splenectomy was indicated. The patient received 5 blood transfusions and on June 29, splenectomy was performed by Dr. Curry. The spleen weighed 890 gm. and the pathologist, Dr. Smith, reported that it showed the characteristics of Banti's syndrome.

Postoperative course was uneventful. The patient was given iron, liver and vitamins. On July 11, Hgb. 65%; red blood cells 3,680,000. July 17, blood platelets 220,800. She was discharged in excellent condition. The remainder of her pregnancy was normal and delivery uncomplicated. She has since had another healthy child and her general condition is very good. November 11, 1947, blood picture Hgb. 80%; red blood cells 4,000,000; white blood cells 12,100; platelets 200,000; bleeding time, normal; reticulocytes, less than 2%.

SUMMARY

The hæmatological disorders in which splenectomy is commonly employed have been mentioned briefly and five cases illustrating its value have been presented.

In considering splenectomy, its indications, limitations and dangers must be fully recognized. The co-operation of the internist, hæmatologist and surgeon is essential. Authorities on the subject appear to be generally agreed that the hæmatological conditions for which the operation may be considered most valuable are as follows: (1) Congenital hæmolytic jaundice. (2) Selected cases of acquired hæmolytic anæmia, acute and chronic forms. (3) Thrombocytopenic purpura, idiopathic or associated with a disease causing hypersplenism. (4) Splenic neutropenia, either primary or symptomatic. (5) Certain cases of Banti's syndrome.

In other disorders of this category splenectomy is either contraindicated or it plays a minor rôle.

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THE ESTIMATION OF HEARING LOSSES FOR PURPOSES OF THE ADJUDICATION OF DISABILITY CLAIMS

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THERE is overwhelming evidence to indicate that the estimation of hearing losses for speech by the use of the conversational voice test, as carried out in any ordinary clinic or office building, is dependent upon so many variable factors as to render it an extremely unreliable test. Among the variables may be mentioned particularly the following:

(*a*) Although examiners endeavour to use residual air only and modulate their voices so as to introduce some degree of accuracy and standardization, it has been shown conclusively that the reflex of raising the voice as distance from the patient increases, is so strong that, experimentally, using a sound level meter and a group of qualified experienced examiners, no change of loudness for the spoken voice at distances of 15 and 5 feet was recorded with the exception of the voices of 3 otologists of the group of examiners used in the experiments (Fowler[1]).

(*b*) Ambient noises are an important factor. The noise level of an ordinary clinic or office building is reliably estimated at from 40 to 65 decibels and for a "sound damped" room from 25 to 35 decibels. Therefore the noise in such environments is sufficient to change the threshold of hearing these amounts without examiners being aware of it.

(*c*) The pitch and clearness of enunciation of the examiner's voice and the acoustics of rooms used are important variables to be considered. A highly pitched voice will show less loss of hearing within the speech range than will a voice of lower pitch. Words used during the test, unless carefully chosen, will give misleading results as there are groups of words heard best within the lower (speech) frequencies and other groups of words containing sounds heard best within frequencies higher than the speech range (for practical purposes the speech range to be regarded as up to 2,500 double vibrations per second). For examples see list of words outlined in publication by Ersner and Sallyman.[2] The conduction and reflection of sound by the interior of rooms have been shown to